Hepatic Arterial Buffer Response: Pathologic Evidence in Non-Cirrhotic Human Liver with Portal Vein Thrombosis

Natalia Rush[1], Hongliu Sun[2], Yukihiro Nakanishi[1], Wadad Mneimneh[1], Paul Y Kwo[3] and Romil Saxena[1]

[1]Department Pathology and Laboratory Medicine, Indiana University, Indianapolis, IN;
[2]Department of Pathology, University of Michigan, Ann Arbor, MI;
[3]Department of Medicine, Indiana University, IN;

Hepatic arterial buffer response (HABR) is the ability of the hepatic artery (HA) to compensate for changes in portal flow. Experimentally, occlusion of the portal vein leads to compensatory increase in HA flow with minimal parenchymal effects. Wether portal vein thrombosis (PVT) causes similar effects in the human liver is unknown. This study aims to answer this question as well as elucidate any microscopic features that may reliably assist diagnosis of PVT in the non-cirrhotic liver. We studied patients with PVT and no concomitant liver pathology. Age and gender matched livers with normal morphology served as controls. Following parameters were graded as subtle or obvious and focal or diffuse in a blinded fashion: sinusoidal dilatation, central and portal vein (PV) dilatation, PV absence, hepatic plate thinning and thickening. Outer and luminal diameters and wall thickness of HA, and outer diameter of accompanying bile ducts (BD) were measured. There were 16 patients (8 men, 8 women; mean age, 46.5 years) who presented with varices (12), ascites (8) and splenomegaly (11). Subtle and or focal dilatations of CV, PV and sinusoids as well as thinning/thickening of hepatic plates were common findings in both groups but were diffuse and obvious predominantly in cases of PVT. Absence or attenuation of PV was seen only in cases of PVT. The large HA were dilated in resection specimens of patients with PVT, $p<0.05$. This difference was not seen in biopsy specimens. There was no difference in the small HA in either biopsy or resection specimens or other measurements of HA or BD. In conclusion, septal branches of the HA dilate as a compensatory response to long standing thrombosis. Microscopic features of PVT are subtle but when obvious and/or diffuse in a patient with non-cirrhotic portal hypertension should raise suspicion for this diagnosis.